Antiinflammatory Activity of the Lapachol and LQB 118 in the Inhaled Bacterial Lipopolysaccharide (LPS)-Induced Lung Inflammation

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The search of new antiinflammatory agents is target of many researches. Natural products and their analogues modified synthetically are molecules of great interest. The aim of our investigation is to study the antiinflammatory effect of the natural product extracted from *Tabebuia*, lapachol, and its synthetic derivative, LQB 118, in the inhaled LPS-induced lung inflammation model, using C57J/BL6 mice. Our results demonstrated that intraperitonial treatment with lapachol (100 mg/Kg) and LQB 118 (10 mg/Kg) reduced neutrophil influx to lungs in mice submitted to inhaled LPS (0,5 mg/mL). Additionally LQB 118 reduced the concentration of the inflammatory mediators TNF-alpha and KC in the bronchoalveolar lavage fluid (BALF). These effects of LQB 118 are comparable to 10 mg/Kg of indomethacin or aspirin, two well-established antiinflammatory drugs. *In vitro,* LQB 118 also inhibited TNF-alpha production in LPS-stimulated mononuclear cells of human peripheral blood (PBMC). The highest concentration tested (100 microM) was able to inhibit almost 100% of TNF-alpha liberation. These data confirm the antiinflammatory action of lapachol observed in a paw edema model (de Almeida, 1990), and reveal the lapachol derivative, LQB 118, as a more potent modulator of inflammation, through the reduction of inflammatory mediators.